ARGinine SPEEDS PULMONARY OXYGEN UPTAKE KINETICS DURING MODERATE-INTENSITY CYCLE EXERCISE

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It has been suggested that nitric oxide (NO) competes with oxygen for binding to cytochrome c oxidase in the electron transport chain and might therefore reversibly inhibit mitochondrial oxygen consumption and slow muscle VO\textsubscript{2} kinetics. Human as well as animal studies that examined the effect of nitro-L-arginine methyl ester (L-NAME) administration (an inhibitor of NO synthesis) on VO\text{2} kinetics led to conflicting results. Some studies reported faster VO\text{2} kinetics during moderate-intensity exercise after L-NAME administration (Kindig et al. 2002; Jones et al. 2003) whilst others found no effect (Grassi et al. 2005). An alternative and opposite approach to further analyse the role of NO in the kinetics of adjustment of oxidative metabolism at exercise onset, is to stimulate NO production by chronic administration of exogenous L-arginine, the substrate for NO synthesis.

Eight physically active males were randomly assigned to receive either placebo (lactose) or L-arginine hydrochloride capsules (3 x 2.42 g/day) for 14 days in a double-blind cross-over design, with a 7 days wash-out period between the two suppletion conditions. On day 11 and day 14 of each condition, the subjects completed two consecutive 6-min bouts of cycle exercise at 80\% of the ventilatory threshold with a 12 min rest interval. VO\text{2} was measured on a breath-by-breath basis and VO\text{2} kinetics were determined with a single exponential model from the averaged data derived from 4 repetitions. Capillary and venous blood samples were taken to determine plasma [La], blood [NH\textsubscript{4}+] and plasma [arginine] respectively. Differences were tested for statistical significance using a 2-tailed paired-samples t-test and a 2 (pre-post) x 2 (placebo-arginine) analysis of variance for repeated measures.

With regard to the pulmonary VO\text{2} kinetics, no significant difference was observed in the time at which the phase II response emerged (mean difference of 1.3 s) or in the phase II amplitude (mean difference of 5.2 ml.min\textsuperscript{-1}) between the two conditions. On the other hand, the time constant was significantly reduced after arginine administration (i.e. $13.9 \pm 3.1$ s vs. $15.8 \pm 2.6$ s in the control condition, $P=0.01$). There were no differences in circulating lactate, NH\textsubscript{4}+ and arginine prior to and during exercise.

The principle finding is that exogenous L-arginine administration speeds the phase II pulmonary VO\text{2} response by 12\% at the onset of moderate-intensity exercise in humans. If the L-arginine administration stimulated the NO production, our results suggest that the down-regulation of mitochondrial respiration by NO does not limit the rate at which pulmonary VO\text{2} rises in the transition from rest to constant-load moderate-intensity exercise.


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